

# **METABOLIC SYNDROME AND METAINFLAMMATION: A REVISIT TO THIS CENTRAL AXIS OF CARDIOVASCULAR DISEASES**

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## **RESUMO**

**INTRODUCTION:** Metabolic Syndrome (MetS) has become a global health concern, and it is often associated with industrialized countries, where people are sedentary and constantly eat fast food and ultra-processed foods. MetS is characterized by metainflammation, that is a result of genetic predisposition combined with changes in the body, such as obesity, insulin resistance (IR), high abdominal circumference, dysfunction in glucose metabolism, and cardiovascular diseases (CVDs), also affecting the liver, brain, and intestine. **OBJECTIVE:** this review aims to elucidate the relationship between metainflammation and MetS and to clarify how this chronic inflammatory state contributes to the onset and progression of cardiovascular diseases. **METHODS:** This is a narrative literature review based on a PubMed search using descriptors related to metabolic syndrome, metainflammation, oxidative stress, and cardiovascular risk. Original articles, systematic reviews, and narrative reviews that evidenced the relationship between metainflammation and metabolic syndrome as potential causes of cardiovascular disease were included. **RESULTS:** MetS, also known as Syndrome X, is associated with obesity, IR, hypertension, elevated levels of triglycerides, and reduced levels of high-density lipoprotein (HDL- c). In patients with MetS, the excess calorie consumption leads to an increase in the amount of visceral white adipose tissue (WAT), which will be responsible for the release of hormones and pro-inflammatory cells that will cause imbalance in glucose metabolism, IR, liver complications, increased release of free fatty acids (FFA) and cholesterol, establishing a condition known as metainflammation. MetS also promotes a shift in adipose tissue function toward a pro-inflammatory profile, with increased release of cytokines and activation of pathways such as Jun N-Terminal Kinase (JNK) and I $\kappa$ B kinase (IKK) that are responsible for the cascade release of pro-inflammatory mediators, in addition to causing IR. M1 macrophages infiltrate adipose tissue and amplify inflammation, contributing to elevated free fatty acids and IR. Interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF- $\alpha$ ), which is released by adipocytes and macrophages, further worsen metabolic dysfunction, promoting endothelial damage and atherosclerosis. These processes reinforce a vicious cycle in which obesity and IR perpetuate each other. The consequences of these changes in the body can lead to lower cognitive performance, vascular dementia, tissue fibrosis, atherogenesis, and other cardiovascular problems. For example, studies have shown that oxidative stress is linked to atrial fibrillation and can also contribute to neuroinflammation. Altogether, these interconnected mechanisms highlight the systemic impact of MetS, extending beyond metabolic dysfunction to multi-organ impairment and increased morbidity. **CONCLUSION:** MetS is driven by a persistent pro-inflammatory and pro-oxidative state that disrupts metabolic homeostasis and promotes endothelial dysfunction, atherogenesis, and systemic complications. This alteration in metabolic homeostasis accelerates cardiovascular damage and highlights the importance of early interventions targeting inflammation and metabolic imbalance.

**Palavras-chave:** Metainflammation Metabolic Syndrome/MetS Cardiovascular Diseases.